CLINICAL UTILITY OF TISSUE DOPPLER IMAGING IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION COMPLICATED BY CARDIOGENIC SHOCK

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CERTIFICATE

This is to certify that the dissertation entitled ***CLINICAL UTILITY OF TISSUE DOPPLER IMAGING IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION COMPLICATED BY CARDIOGENIC SHOCK*** is the bonafide original work of **DR.G. KANNAPPAN** in partial fulfillment of the requirements for D.M. Branch-II (CARDIOLOGY) examination of THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY to be held in August 2010.The period of post-graduate study and training was from August 2007 to July 2010.

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DECLARATION

I, DR.G.Kannappan solemnly declare that this dissertation entitled **"CLINICAL UTILITY OF TISSUE DOPPLER IMAGING IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION COMPLICATED BY CARDIOGENIC SHOCK"** is a bonafide work done by me at the department of Cardiology, Government Stanley Medical College and Hospital during the period 2007 – 2010 under the guidance and supervision of the Professor and Head of the department of Cardiology of Government Stanley Medical College and Hospital Professor **Dr**.**G.KARTHIKEYAN.MD.DM**. This dissertation is submitted to The Tamil Nadu Dr.M.G.R Medical University, towards partial fulfillment of requirement for the award of **D.M. Degree (Branch-II) in Cardiology.**

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CLINICAL UTILITY OF TISSUE DOPPLER IMAGING IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION COMPLICATED BY CARDIOGENIC SHOCK

CONTENTS

INTRODUCTION

Cardiogenic shock (CS) is a state of inadequate tissue perfusion due to cardiac dysfunction.Cardiogenic shock is the most severe clinical expression of left ventricular failure and is associated with extensive damage to the left ventricular myocardium in more then 80% of STEMI patients in whom it occurs : the reminder have a mechanical defect such as ventricular septal or papillary muscle rupture or predominant right ventricular infarction .

In the past cardiogenic shock has been reported to occur in up to 20 % of patients with STEMI,but estimates from recent large trails and observational databases report an incidence rate in the range of 7%. This low output state is characterized by elevated ventricular filling pressures, low cadiac output, systemic hypotension and evidence of vital organ hypoperfusion .

Between 1995 and 2004, the National Registry of Myocardial Infarction database recorded 300 000 ST elevation MI's of which 8.6% presented with Cardiogenic shock.. In patients hospitalized for either myocardial infarction, decompensated heart failure or following cardiac surgery, Cardiogenic shock was the leading cause of death resulting in mortality rates of up to 80% . Although rapid stabilization and treatment of reversible causes via early revascularization is a priority, the mortality rate due to Cardiogenic shock in the current era remains high. Of note, although the incidence of cardiogenic shock in patients with STEMI has been relatively stable since the mid -1970s, the short –term mortality rate has has decreased from 70 to 80 % in the 1970s to 50 to 60 % in the 1990s.Cardiogenic shock is the cause of death in about 60% of patients dying after fibrinolysis for STEMI.

Prognostic echocardiographic factors for determination of early recovery after Cardiogenic shock are limited. Short and long term mortality appears to be associated with initial depressed LV systolic function and mitral

regurgitation (MR) as assessed by echocardiography. In patients with LV ejection fraction (EF) less than 30%, survival at one year was 24% versus 56% for those with preserved systolic function.

Tissue Doppler imaging (TDI) is a sensitive, noninvasive echocardiographic method that records velocity of tissue motion within the myocardium. TDI has been evaluated in both in vitro and in vivo studies, allowing for the quantitative assessment of both global and regional function of the myocardium. Indices derived from TDI, including systolic velocity (S'), early (E') and late (A') diastolic velocities of the mitral annulus, are reduced in heart failure patients (EF < 30%) and portend a poor prognosis. Transmitral to early diastolic velocity ratio (E/E') obtained via TDI correlates strongly with LV filling pressures. An E/E' ratio > 10 identified a pulmonary capillary wedge pressure PCWP > 15 mm Hg with a sensitivity of 92% and a specificity of 80%.

The objective of this study was to describe TDI derived indices in patients presenting with cardiogenic shock after acute myocardial infarction but prior to percutaneous revascularization.

AIM OF THE STUDY

Echocardiography is widely used in the management of patients with cardiogenic shock (CS). Left ventricular ejection fraction (EF) has been shown to be an independent predictor of survival in CS. Tissue Doppler Imaging (TDI) is a sensitive echocardiographic technique that allows for the early quantitative assessment of regional left ventricular dysfunction. TDI derived indices, including systolic velocity (S'), early (E') and late (A') diastolic velocities of the mitral annulus, are reduced in heart failure patients and portend a poor prognosis. In addition to chronic CHF, a higher E/E' has also been shown to correlate with a worse prognosis in acute myocardial infarction and cardiogenic shock.

The early diastolic velocity of the mitral valve annulus (e') reflects the rate of myocardial relaxation. When combined with measurement of the early transmitral flow velocity (E), the resultant ratio (E/e') correlates well with mean LVDP. An E/e' ratio >15 is an excellent predictor of an elevated mean LVDP and would predict poorer survival after acute MI.

The objective of this study was to analyse and describe TDI derived indices in patients presenting with cardiogenic shock after acute myocardial infarction and compare to patients with chronic CHF and there by to assess the prognostic value of a noninvasive measure of left ventricular diastolic pressure early after acute myocardial infarction (MI).

REVIEW OF LITERATURE

Echocardiography is now the most commonly used noninvasive tool for the assessment of cardiac anatomy and function. In addition to commonly established roles such as confirming diagnosis, etiologic work-up, complication screening, and disease monitoring, echocardiography plays an important clinical role in prognostic assessment. Conventional echocardiographic predictors of poor outcome, such as left ventricular ejection fraction and restrictive filling pattern have recently been supplemented by tissue Doppler imaging .

Disadvantages of Conventional Echo Imaging

Endocardial border is not clearly defined in some pts

Data is dependant on reflected signal amplitude

Provides only semiquantative data

Inter-observer/Intra-observer variation

Limited ability to detect subtle changes

Human eye cannot reliably visualize time events occurring in < 90 msec for single image.

Tissue Doppler imaging (TDI) is evolving as a useful echocardiographic tool for quantitative assessment of left ventricular (LV) systolic and diastolic function. Recent studies have explored the prognostic role of TDI-derived parameters in major cardiac diseases, such as heart failure, acute myocardial infarction, and hypertension. In these conditions, myocardial mitral annular (Sa) systolic and early diastolic (Ea) velocities have been shown to predict mortality or cardiovascular events.. In heart failure and after myocardial

infarction, noninvasive assessment of LV diastolic pressure by transmitral to mitral annular early diastolic velocity ratio (E/Ea) is a strong prognosticator, especially when E/Ea is >15. In addition, systolic intraventricular dyssynchrony measured by segmental analysis of myocardial velocities is another independent predictor of adverse clinical outcome in heart failure subjects, even when the QRS duration is normal. In heart failure patients who received cardiac resynchronization therapy, the presence of systolic dyssynchrony at baseline is associated with favorable LV remodeling, which in turn predicts a favorable long-term clinical outcome. Finally, TDI and derived deformation parameters improve prognostic assessment during dobutamine stress echocardiography.

Tissue Doppler imaging is a robust and reproducible echocardiographic tool which has permitted a quantitative assessment of both global and regional function and timing of myocardial events. In clinical practice, the myocardial time-velocity curve can be reconstructed either on line as spectral pulsed TDI or off line from 2-dimensional color-coded TDI image loops. It is important to recognize that myocardial velocities obtained from the on-line spectral pulsed TDI curve are higher than those reconstructed off-line from 2D color-coded TDI images. Most published studies have examined the long-axis function of the heart by TDI from apical views.

A number of parameters from TDI have been proposed to be useful in various cardiac diseases. In systole, potentially important prognosticators of TDI include peak systolic velocity in ejection period measured at mitral annulus (Sa) as well as systolic dyssynchrony assessment. In diastole, potentially important prognosticators include peak myocardial early diastolic velocity measured at the mitral annulus (Ea) as well as measurement of transmitral to TDI early diastolic velocity ratio (E/Ea). These myocardial velocity measurements with TDI have been shown to be useful in various diseases and in patients undergoing stress echocardiography for suspected coronary heart disease.

PHYSIOLOGY

The optimal performance of the left ventricle depends on its ability to cycle between two states: (1) a compliant chamber in diastole that allows the left ventricle to fill from low LA pressure and (2) a stiff chamber (rapidly rising pressure) in systole that ejects the stroke volume at arterial pressures. The ventricle has two alternating functions: systolic ejection and diastolic filling. Furthermore, the stroke volume must increase in response to demand, such as exercise, without much increase in LA pressure. The theoretically optimal LV pressure curve is rectangular, with an instantaneous rise to peak and an instantaneous fall to low diastolic pressures, which allows for the maximum time for LV filling. This theoretically optimal situation is approached by the cyclic interaction of myofilaments and assumes competent mitral and aortic valves. Diastole starts at aortic valve closure and includes LV pressure fall, rapid filling, diastasis and atrial contraction.

Elevated filling pressures are the main physiologic consequence of diastolic dysfunction. Filling pressures are considered elevated when the mean pulmonary capillary wedge pressure (PCWP) is >12 mm Hg or when the LVEDP is >16 mm Hg. Filling pressures change minimally with exercise in healthy subjects. Exercise-induced elevation of filling pressures limits exercise capacity and can indicate diastolic dysfunction. LV filling pressures are determined mainly by filling and passive properties of the LV wall but may be further modulated by incomplete myocardial relaxation and variations in diastolic myocardial tone.

At the molecular level, the cyclic interaction of myofilaments leads to a muscular contraction and relaxation cycle. Relaxation is the process whereby the myocardium returns after contraction to its unstressed length and force. In normal hearts, and with normal load, myocardial relaxation is nearly complete at minimal LV pressure. Contraction and relaxation belong to the same

molecular processes of transient activation of the myocyte and are closely intertwined. Relaxation is subjected to control by load, inactivation, and asynchrony.

Increased afterload or late systolic load will delay myocardial relaxation, especially when combined with elevated preload, thereby contributing to elevating filling pressures. Myocardial inactivation relates to the processes underlying calcium extrusion from the cytosol and cross-bridge detachment and is affected by a number of proteins that regulate calcium homeostasis, cross-bridge cycling, and energetics. Minor regional variation of the timing of regional contraction and relaxation is physiological. However, dyssynchronous relaxation results in a deleterious interaction between early reextension in some segments and postsystolic shortening of other segments and contributes to delayed global LV relaxation and elevated filling pressures.

Tau is a widely accepted invasive measure of the rate of LV relaxation, which will be 97% complete at a time corresponding to 3.5 t after dP/dt_{min}. Diastolic dysfunction is present when $t > 48$ ms. In addition, the rate of relaxation may be evaluated in terms of LV dP/dt_{min} and indirectly with the isovolumetric relaxation time (IVRT), the time interval between aortic valve closure and mitral valve opening.

LV filling is determined by the interplay between LV filling pressures and filling properties. These filling properties are described with stiffness or inversely with compliance and commonly refer to end-diastolic properties. Several factors extrinsic and intrinsic to the left ventricle determine these end-diastolic properties. Extrinsic factors are mainly pericardial restraint and ventricular interaction. Intrinsic factors include myocardial stiffness (cardiomyocytes and extracellular matrix), myocardial tone, chamber geometry and wall thickness.

A distinct aspect of diastolic function is related to longitudinal function and torsion. *Torrent-Guasp et al* described how the ventricles may to some extent be assimilated to a single myofiber band starting at the right ventricle below the pulmonary valve and forming a double helix extending to the left ventricle, where it attaches to the aorta. This double helicoidal fiber orientation leads to systolic twisting (torsion) and diastolic untwisting (torsional recoil).

KEY POINTS

1 Diastolic function is related to myocardial relaxation and passive LV properties and is modulated by myocardial tone.

2 Myocardial relaxation is determined by load, inactivation, and nonuniformity.

3 Myocardial stiffness is determined by the myocardial cell (eg, titin) and by the interstitial matrix (fibrosis).

TISSUE DOPPLER ANNULAR EARLY AND LATE DIASTOLIC VELOCITIES

A ACQUISITION AND FEASIBILITY

PW tissue Doppler imaging (DTI) is performed in the apical views to acquire mitral annular velocities. Although annular velocities can also be obtained by color-coded DTI, this method is not recommended, because the validation studies were performed using PW Doppler. The sample volume should be positioned at or 1 cm within the septal and lateral insertion sites of the mitral leaflets and adjusted as necessary usually 5-10 mm to cover the longitudinal excursion of the mitral annulus in both systole and diastole. Attention should be directed to Doppler spectral gain settings, because annular velocities have high signal amplitude. Most current ultrasound systems have tissue Doppler presets for the proper velocity scale and Doppler wall filter

settings to display the annular velocities. In general, the velocity scale should be set at about 20 cm/s above and below the zero-velocity baseline, though lower settings may be needed when there is severe LV dysfunction and annular velocities are markedly reduced (scale set to 10-15 cm/s). Minimal angulation (<20°) should be present between the ultrasound beam and the plane of cardiac motion. DTI waveforms can be obtained in nearly all patients (>95%), regardless of 2D image quality. It is recommended that spectral recordings be obtained at a sweep speed of 50 to 100 mm/s at end-expiration and that measurements should reflect the average of ≥3 consecutive cardiac cycles.

B MEASUREMENTS

Primary measurements include the systolic (S), early diastolic, and late diastolic velocities. The early diastolic annular velocity has been expressed as Ea, Em, E′, or e′, and the late diastolic velocity as Aa, Am, A′, or a′. The writing group favors the use of e′ and a′, because Ea is commonly used to refer to arterial elastance. The measurement of e′ acceleration and DT intervals, as well as acceleration and deceleration rates, does not appear to contain incremental information to peak velocity alone and need not be performed routinely. On the other hand, the time interval between the QRS complex and e′ onset is prolonged with impaired LV relaxation and can provide incremental information in special patient populations. For the assessment of global LV diastolic function, it is recommended to acquire and measure tissue Doppler signals at least at the septal and lateral sides of the mitral annulus and their average, given the influence of regional function on these velocities and time intervals.

Once mitral flow, annular velocities, and time intervals are acquired, it is possible to compute additional time intervals and ratios. The ratios include annular e′/a′ and the mitral inflow E velocity to tissue Doppler e′ (E/e′) ratio. The latter ratio plays an important role in the estimation of LV filling pressures.

For time intervals, the time interval between the QRS complex and the onset of mitral E velocity is subtracted from the time interval between the QRS complex and e′ onset to derive ($T_{E\text{-}e'}$), which can provide incremental information to E/e′ in special populations. Technically, it is important to match the RR intervals for measuring both time intervals time to E and time to e′ and to optimize gain and filter settings, because higher gain and filters can preclude the correct identification of the onset of e′ velocity.

C HEMODYNAMIC DETERMINANTS

The hemodynamic determinants of e′ velocity include LV relaxation preload, systolic function, and LV minimal pressure. A significant association between e′ and LV relaxation was observed in animal and human studies. For preload, LV filling pressures have a minimal effect on e′ in the presence of impaired LV relaxation.

On the other hand, with normal or enhanced LV relaxation, preload increases e′. Therefore, in patients with cardiac disease, e′ velocity can be used to correct for the effect of LV relaxation on mitral E velocity, and the E/e′ ratio can be applied for the prediction of LV filling pressures. The main hemodynamic determinants of a′ include LA systolic function and LVEDP, such that an increase in LA contractility leads to increased a′ velocity, whereas an increase in LVEDP leads to a decrease in a’

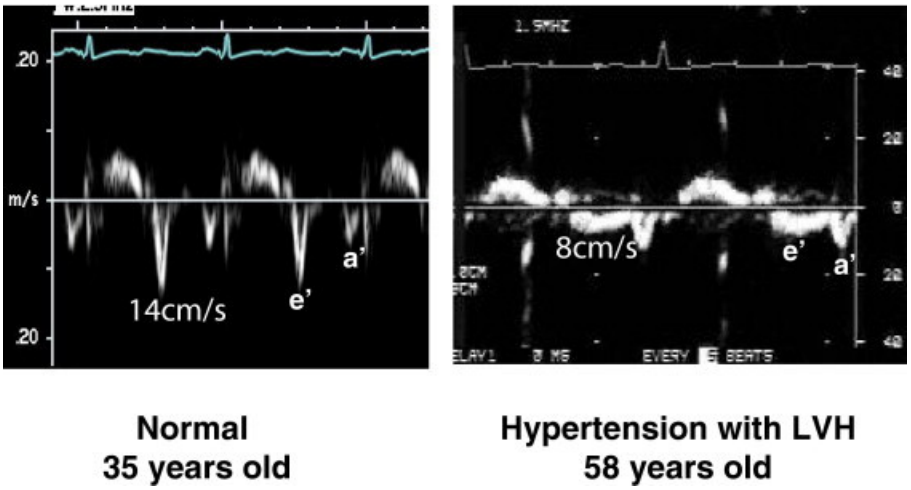


Figure 1 Tissue Doppler recording from the lateral mitral annulus from a normal subject aged 35 years *(left)* (e′ = 14 cm/s) and a 58-year-old patient with hypertension, LV hypertrophy, and impaired LV relaxation *(right)* (e′ = 8 cm/s).

In the presence of impaired LV relaxation and irrespective of LA pressure, the e′ velocity is reduced and delayed, such that it occurs at the LA-LV pressure crossover point. On the other hand, mitral E velocity occurs earlier with pseudonormal LV filling or restrictive LV filling. Accordingly, the time interval between the onset of E and e′ is prolonged with diastolic dysfunction. Animal and human studies have shown that ($T_{E\text{-}e'}$) is strongly dependent on the time constant of LV relaxation and LV minimal pressure.

D NORMAL VALUES

Normal values of DTI-derived velocities are influenced by age, similar to other indices of LV diastolic function. With age, e′ velocity decreases, whereas a′ velocity and the E/e′ ratio increase.

Table 1 -- *Normal values for Doppler-derived diastolic measurements*

Measurement	Age group (y)			
	16-20	21-40	41-60	>60
IVRT (ms)	50 ± 9 (32-68)	67 ± 8 (51-83)	74 ± 7 (60-88)	87 ± 7 (73-101)
E/A ratio	1.88 ± 0.45 (0.98-2.78)	1.53 ± 0.40 (0.73-2.33)	1.28 ± 0.25 (0.78-1.78)	0.96 ± 0.18 (0.6-1.32)
DT (ms)	142 ± 19 (104-180)	166 ± 14 (138-194)	181 ± 19 (143-219)	200 ± 29 (142-258)
A duration (ms)	113 ± 17 (79-147)	127 ± 13 (101-153)	133 ± 13 (107-159)	138 ± 19 (100-176)
PV S/D ratio	0.82 ± 0.18 (0.46-1.18)	0.98 ± 0.32 (0.34-1.62)	1.21 ± 0.2 (0.81-1.61)	1.39 ± 0.47 (0.45-2.33)
PV Ar (cm/s)	16 ± 10 (1-36)	21 ± 8 (5-37)	23 ± 3 (17-29)	25 ± 9 (11-39)
PV Ar duration (ms)	66 ± 39 (1-144)	96 ± 33 (30-162)	112 ± 15 (82-142)	113 ± 30 (53-173)
Septal e′ (cm/s)	14.9 ± 2.4 (10.1-19.7)	15.5 ± 2.7 (10.1-20.9)	12.2 ± 2.3 (7.6-16.8)	10.4 ± 2.1 (6.2-14.6)
Septal e′/a′ ratio	2.4	1.6 ± 0.5 (0.6-2.6)	1.1 ± 0.3 (0.5-1.7)	0.85 ± 0.2 (0.45-1.25)
Lateral e′ (cm/s)	20.6 ± 3.8 (13-28.2)	19.8 ± 2.9 (14-25.6)	16.1 ± 2.3 (11.5-20.7)	12.9 ± 3.5 (5.9-19.9)
Lateral e′/a′ ratio	3.1	1.9 ± 0.6 (0.7-3.1)	1.5 ± 0.5 (0.5-2.5)	0.9 ± 0.4 (0.1-1.7)

Data are expressed as mean ± SD (95% confidence interval). Note that for e′ velocity in subjects aged 16 to 20 years, values overlap with those for subjects aged 21 to 40 years. This is because e′ increases progressively with age in children and adolescents. Therefore, the e′ velocity is higher in a normal

20-year-old than in a normal 16-year-old, which results in a somewhat lower average e′ value when subjects aged 16 to 20 years are considered.

E CLINICAL APPLICATION

Mitral annular velocities can be used to draw inferences about LV relaxation and along with mitral peak E velocity (E/e′ ratio) can be used to predict LV filling pressures. To arrive at reliable conclusions, it is important to take into consideration the age of a given patient, the presence or absence of cardiovascular disease, and other abnormalities noted in the echocardiogram. Therefore, e′ and the E/e′ ratio are important variables but should not be used as the sole data in drawing conclusions about LV diastolic function.

It is preferable to use the average e′ velocity obtained from the septal and lateral sides of the mitral annulus for the prediction of LV filling pressures. Because septal e′ is usually lower than lateral e′ velocity, the E/e′ ratio using septal signals is usually higher than the ratio derived by lateral e′, and different cutoff values should be applied on the basis of LVEF, as well as e′ location. Although single-site measurements are sometimes used in patients with globally normal or abnormal LV systolic function, it is imperative to use the average septal and lateral e′ velocity in the presence of regional dysfunction. Additionally, it is useful to consider the range in which the ratio falls. Using the septal E/e′ ratio, a ratio < 8 is usually associated with normal LV filling pressures, whereas a ratio > 15 is associated with increased filling pressures. When the value is between 8 and 15, other echocardiographic indices should be used. A number of recent studies have noted that in patients with normal EFs, lateral tissue Doppler signals (E/e′ and e′/a′) have the best correlations with LV filling pressures and invasive indices of LV stiffness. These studies favor the use of lateral tissue Doppler signals in this population.

$T_{E\text{-}e'}$ is particularly useful in situations in which the peak e′ velocity has its limitations, and the average of 4 annular sites is more accurate than a single

site measurement for this time interval. The clinical settings in which it becomes advantageous to use it include subjects with normal cardiac function or those with mitral valve disease and when the E/e′ ratio is 8 to 15. In particular, an IVRT/$T_{E\text{-}e'}$ ratio < 2 has reasonable accuracy in identifying patients with increased LV filling pressures.

F LIMITATIONS

There are both technical and clinical limitations. For technical limitations, proper attention to the location of the sample size, as well as gain, filter, and minimal angulation with annular motion, is essential for reliable velocity measurements. With experience, these are highly reproducible with low variability. Because time interval measurements are performed from different cardiac cycles, additional variability is introduced. This limits their application to selective clinical settings in which other Doppler measurements are not reliable.

There are a number of clinical settings in which annular velocity measurements and the E/e′ ratio should not be used. In normal subjects, e′ velocity is positively related to preload and the E/e′ ratio may not provide a reliable estimate of filling pressures. These individuals can be recognized by history, normal cardiac structure and function, and the earlier or simultaneous onset of annular e′ in comparison with mitral E velocity. Additionally, e′ velocity is usually reduced in patients with significant annular calcification, surgical rings, mitral stenosis, and prosthetic mitral valves. It is increased in patients with moderate to severe primary MR and normal LV relaxation due to increased flow across the regurgitant valve. In these patients, the E/e′ ratio should not be used, but the IVRT/$T_{E\text{-}e'}$ ratio can be applied.

Patients with constrictive pericarditis usually have increased septal e′, due largely to preserved LV longitudinal expansion compensating for the limited lateral and anteroposterior diastolic excursion. Lateral e′ may be less

than septal e′ in this condition, and the E/e′ ratio was shown to relate inversely to LV filling pressures or annulus paradoxus.

KEY POINTS

1 PW DTI is performed in the apical views to acquire mitral annular velocities.

2 The sample volume should be positioned at or 1 cm within the septal and lateral insertion sites of the mitral leaflets.

3 It is recommended that spectral recordings be obtained at a sweep speed of 50 to 100 mm/s at end-expiration and that measurements should reflect the average of ≥3 consecutive cardiac cycles.

4 Primary measurements include the systolic and early (e′) and late (a′) diastolic velocities.

5 For the assessment of global LV diastolic function, it is recommended to acquire and measure tissue Doppler signals at least at the septal and lateral sides of the mitral annulus and their average.

6 In patients with cardiac disease, e′ can be used to correct for the effect of LV relaxation on mitral E velocity, and the E/e′ ratio can be applied for the prediction of LV filling pressures.

7 The E/e′ ratio is not accurate as an index of filling pressures in patients with heavy annular calcification, mitral valve disease, and constrictive pericarditis

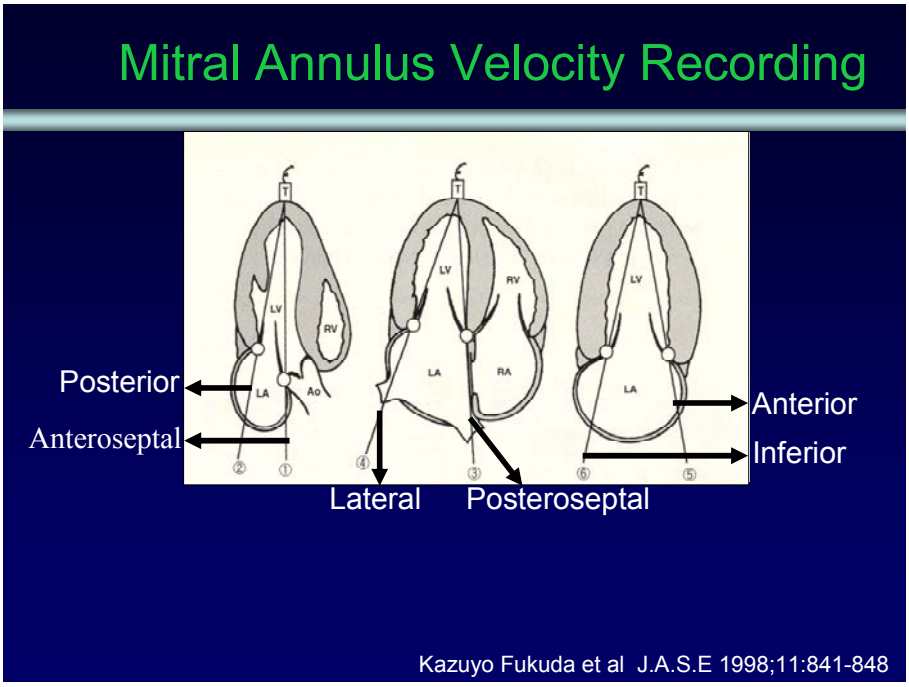
TDI -CLINICAL APPLICATIONS

- Assessment of global& regional LV systolic & diastolic dysfunction
- Quantification of regional wall motion
- Assessment of Left Ventricular Filling Pressure
- In Myocardial Ischemia, Infarction
- In Myocardial Viability
- During stress echocardiography
- Differentiation of restrictive cardiomyopathy & constrictive pericarditis
- Heart failure & after cardiac transplantation
- To differentiate pathological from physiological LV hypertrophy.
- Sub clinical Left Ventricular Dysfunction- RHD, Drug induced
- To asses RV function

TDI –ASSESSMENT OF GLOBAL SYSTOLIC FUNCTION

- Average of 6 mitral annular velocities around mitral annulus

FIGURE 2



- Normal E' at medial annulus > 10 cm/s, lateral annulus >15 cm/sec
- Peak systolic velocity of <7.5cm/sec correlates with an EF of <50% with sensitivity 79% & specificity of 88%
- Tricuspid systolic annular velocity < 11.5 cm/s predicted RVEF <45% with sensitivity of 90% &specificity of 85%

DIASTOLIC STRESS TEST

Many patients with diastolic dysfunction have symptoms, mainly with exertion, because of the rise in filling pressures that is needed to maintain adequate LV filling and stroke volume. Therefore, it is useful to evaluate LV filling pressure with exercise as well, similar to the use of exercise to evaluate patients with coronary artery or mitral valve disease. The E/e′ ratio has been applied for that objective. In subjects with normal myocardial relaxation, E and e′ velocities increase proportionally, and the E/e′ ratio remains unchanged or is reduced.

However, in patients with impaired myocardial relaxation, the increase in e′ with exercise is much less than that of mitral E velocity, such that the E/e′ ratio increases. In that regard, E/e′ was shown to relate significantly to LV filling pressures during exercise, when Doppler echocardiography was acquired simultaneously with cardiac catheterization. In addition, mitral DT decreases slightly in normal individuals with exercise, but shortens > 50 ms in patients with a marked elevation of filling pressures.

Table 2 -- Changes in mitral and tissue Doppler septal velocities with exercise in normal subjects

Variable	Baseline	Exercise
E (cm/s)	73 ± 19	90 ± 25
A (cm/s)	69 ± 17	87 ± 22
DT (ms)	192 ± 40	176 ± 42
e′ (cm/s)	12 ± 4	15 ± 5
E/e′	6.7 ± 2.2	6.6 ± 2.5

In cardiac patients, mitral E velocity increases with exertion and stays increased for a few minutes after the termination of exercise, whereas e′ velocity remains reduced at baseline, exercise, and recovery. Therefore, E and e′ velocities can be recorded after exercise, after 2D images have been obtained for wall motion analysis. Furthermore, the delayed recording of Doppler

velocities avoids the merging of E and A velocities that occurs at faster heart rates. Exercise is usually performed using a supine bicycle protocol, and TR signals by CW Doppler are recorded as well to allow for the estimation of PA systolic pressure at rest and during exercise and recovery. Diastolic stress echocardiography has been also performed with dobutamine infusion, and restrictive filling with dobutamine was shown to provide prognostic information.

The test is most useful in patients with unexplained exertional dyspnea who have mild diastolic dysfunction and normal filling pressures at rest. However, the paucity of clinical data and the potential limitations in patients with regional LV dysfunction, mitral valve disease, and atrial fibrillation preclude recommendations for its routine clinical use at this time.

MITRAL INFLOW

A. ACQUISITION AND FEASIBILITY

Pulsed-wave (PW) Doppler is performed in the apical 4-chamber view to obtain mitral inflow velocities to assess LV filling. Color flow imaging can be helpful for optimal alignment of the Doppler beam, particularly when the left ventricle is dilated. Performing CW Doppler to assess peak E (early diastolic) and A (late diastolic) velocities should be performed before applying the PW technique to ensure that maximal velocities are obtained. A 1-mm to 3-mm sample volume is then placed between the mitral leaflet tips during diastole to record a crisp velocity profile. Optimizing spectral gain and wall filter settings is important to clearly display the onset and cessation of LV inflow. Excellent-quality mitral inflow waveforms can be recorded in nearly all patients. Spectral mitral velocity recordings should be initially obtained at sweep speeds of 25 to 50 mm/s for the evaluation of respiratory variation of flow velocities, as seen in patients with pulmonary or pericardial disease . If

variation is not present, the sweep speed is increased to 100 mm/s, at end-expiration, and averaged over 3 consecutive cardiac cycles.

B MEASUREMENTS

Primary measurements of mitral inflow include the peak early filling (E-wave) and late diastolic filling (A-wave) velocities, the E/A ratio, deceleration time (DT) of early filling velocity, and the IVRT, derived by placing the cursor of CW Doppler in the LV outflow tract to simultaneously display the end of aortic ejection and the onset of mitral inflow. Secondary measurements include mitral A-wave duration obtained at the level of the mitral annulus, diastolic filling time, the A-wave velocity-time integral, and the total mitral inflow velocity-time integral with the sample volume at the level of the mitral annulus. Middiastolic flow is an important signal to recognize. Low velocities can occur in normal subjects, but when increased ≥20 cm/s, they often represent markedly delayed LV relaxation and elevated filling pressures.

C NORMAL VALUES

Age is a primary consideration when defining normal values of mitral inflow velocities and time intervals. With increasing age, the mitral E velocity and E/A ratio decrease, whereas DT and A velocity increase. Normal values are shown in Table 1.

A number of variables other than LV diastolic function and filling pressures affect mitral inflow, including heart rate and rhythm, PR interval, cardiac output, mitral annular size, and LA function. Age-related changes in diastolic function parameters may represent a slowing of myocardial relaxation, which predisposes older individuals to the development of diastolic heart failure.

D INFLOW PATTERNS AND HEMODYNAMICS

Mitral inflow patterns are identified by the mitral E/A ratio and DT. They include normal, impaired LV relaxation, pseudonormal LV filling (PNF), and restrictive LV filling. The determination of PNF may be difficult by mitral inflow velocities alone. Additionally, less typical patterns are sometimes observed, such as the triphasic mitral flow velocity flow pattern. The most abnormal diastolic physiology and LV filling pattern variants are frequently seen in elderly patients with severe and long-standing hypertension or patients with Hypertrophic cardiomyopathy.

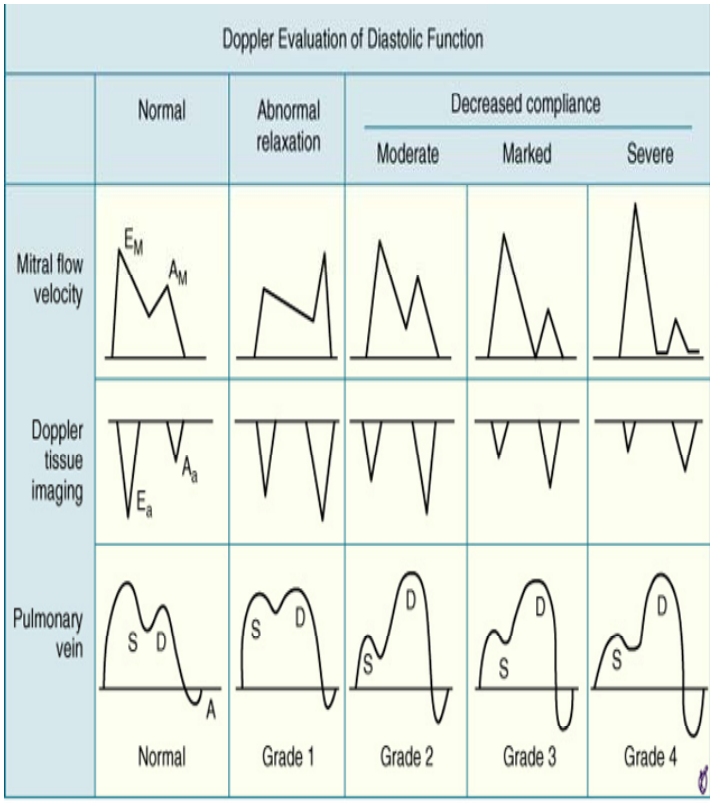


FIGURE 3 Schematic representation of mitral inflow, Doppler tissue imaging of the annulus, and pulmonary vein flow in normal and abnormal diastolic states. Normal mitral inflow is biphasic and consists of an early velocity (Em) and a late flow velocity related to atrial contraction (Am). Doppler tissue

imaging of the annulus results in a similar pattern of early and late (Ea and Aa) annular velocities opposite in direction to the mitral inflow velocity. In patients with normal diastolic function, both Em and Ea exceed Am and Aa. In disease-free states, pulmonary vein flow is multiphasic, with roughly equal systolic and diastolic forward flow and a relatively narrow low velocity retrograde pulmonary vein a-wave. With varying degrees of diastolic dysfunction, there are predictable changes in mitral flow velocity, Doppler tissue annular velocities, and pulmonary vein velocities as noted in the schematic.

It is well established that the mitral E-wave velocity primarily reflects the LA-LV pressure gradient during early diastole and is therefore affected by preload and alterations in LV relaxation. The mitral A-wave velocity reflects the LA-LV pressure gradient during late diastole, which is affected by LV compliance and LA contractile function. E-wave DT is influenced by LV relaxation, LV diastolic pressures following mitral valve opening, and LV compliance (ie, the relationship between LV pressure and volume). Alterations in LV end-systolic and/or end-diastolic volumes, LV elastic recoil, and/or LV diastolic pressures directly affect the mitral inflow velocities (ie, E wave) and time intervals (ie, DT and IVRT).

E CLINICAL APPLICATION TO PATIENTS WITH DEPRESSED AND NORMAL EFS

In patients with dilated cardiomyopathies, PW Doppler mitral flow velocity variables and filling patterns correlate better with cardiac filling pressures, functional class, and prognosis than LVEF. Patients with impaired LV relaxation filling are the least symptomatic, while a short IVRT, short mitral DT, and increased E/A velocity ratio characterize advanced diastolic dysfunction, increased LA pressure, and worse functional class. A restrictive filling pattern is associated with a poor prognosis, especially if it persists after preload reduction. *Likewise, a pseudonormal or restrictive filling pattern*

associated with acute myocardial infarction indicates an increased risk for heart failure, unfavorable LV remodeling, and increased cardiovascular mortality, irrespective of EF.

In patients with coronary artery disease or hypertrophic cardiomyopathy, in whom LV EFs are ≥50%, mitral variables correlate poorly with hemodynamics. This may be related to the marked variation in the extent of delayed LV relaxation seen in these patients, which may produce variable transmitral pressure gradients for similar LA pressures. A restrictive filling pattern and LA enlargement in a patient with a normal EF are associated with a poor prognosis similar to that of a restrictive pattern in dilated cardiomyopathy. This is most commonly seen in restrictive cardiomyopathies, especially amyloidosis, and in heart transplant recipients.

Key Points

1 PW Doppler is performed in the apical 4-chamber view to obtain mitral inflow velocities to assess LV filling.

2 A 1-mm to 3-mm sample volume is then placed between the mitral leaflet tips during diastole to record a crisp velocity profile.

3 Primary measurements include peak E and A velocities, E/A ratio, DT, and IVRT.

4 Mitral inflow patterns include normal, impaired LV relaxation, PNF, and restrictive LVfilling.

5 In patients with dilated cardiomyopathies, filling patterns correlate better with filling pressures, functional class, and prognosis than LVEF.

6 In patients with coronary artery disease and those with hypertrophic cardiomyopathy in whom the LV EFs are ≥50%, mitral velocities correlate poorly with hemodynamics.

ESTIMATION OF LV FILLING PRESSURES IN PATIENTS WITH DEPRESSED EFS

The mitral inflow pattern by itself can be used to estimate filling pressures with reasonable accuracy in this population. Furthermore, the changes in the inflow pattern can be used to track filling pressures in response to medical therapy. In patients with impaired relaxation patterns and peak E velocities < 50 cm/s, LV filling pressures are usually normal. With restrictive filling, mean LA pressure is increased.

The use of additional Doppler parameters is recommended in patients with E/A ratios ≥1 to <2. A change in E/A ratio with the Valsalva maneuver of ≥0.5, a systolic peak velocity/diastolic peak velocity ratio in pulmonary venous flow < 1, Ar − A duration ≥ 30 ms, E/Vp ≥ 2.5, E/e′ (using average e′) ≥ 15, IVRT/$T_{E\text{-}e'}$ < 2, and PA systolic pressure ≥ 35 mm Hg (in the absence of pulmonary disease) can be used to infer the presence of increased filling pressures. Conversely, a change in E/A ratio with the Valsalva maneuver of <0.5, a systolic peak velocity/diastolic peak velocity ratio in pulmonary venous flow > 1, Ar − A duration < 0 ms, E/Vp < 1.4, E/e′ (using average e′) < 8, IVRT/$T_{E\text{-}e'}$ > 2, and PA systolic pressure < 30 mm Hg occur with normal filling pressures. In patients with pseudonormal filling, it is preferable to base the conclusions on ≥2 Doppler findings, giving more weight to signals with higher technical quality. Some LA dilatation commonly occurs in this population, even when LV filling pressures are normal, and therefore should not be used as the final arbitrator in this setting.

TABLE 3 SUMMARY OF STUDIES WHICH ASSESSED THE PROGNOSTIC IMPORTANCE OF TISSUE DOPPLER IMAGING (TDI) PARAMETERS IN CARDIAC DISEASES

Author	Parameters	Disease Group	Sample Size	Duration	End Point	Predictors of Event
Resting echocardiography with TDI						
Wang et al	Mean Sm, Em, Am, from 4 basal LV segments	Various Heart diseases	353 patients 165 controls	23 months	Mortality	Sm<3cm/s Em <3cm/s, Am <4cm/s
Richartz et al	Sa and Ea at septal and lateral mitral annulus	HF	40 patients 25 controls	Cross sectional study	Acute Pulmonary edema	Ea
Wang et al	Mean Sm, Em, Am	HF	182 patients	48 months	Cardiac mortality	Sm, Em, Am, E/Em
Dokainish et al	E/Ea	HF	116 patients	18 months	Cardiac mortality or HF hospitalization	E/Ea >15
Yamamoto et al	E/Ea, Aa	HF	96 patients	29 months	Cardiac mortality or HF hospitalization	E/Ea >15, Aa 5cm/s
Hillis et al.	E/Ea	AMI	250 patients	13 months	Mortality	E/Ea >15
Wang et al	Sm. Em, Am	Hypertention	174 patients	19 months	Cardiac mortality	Sm, Em, Am

AMI = acute myocardial infarction; CHD = coronary heart disease; CRT = cardiac resynchronization therapy; DSE = dobutamine stress echocardiography; HF = heart failure; IVSd = thickness of interventricular septum at end-diastole; LV = left ventricular; Ts-diff = maximum difference in time to peak systolic velocity; Ts-SD = standard deviation of time to peak systolic velocity

TABLE 4 POTENTIALLY USEFUL PARAMETERS OF SYSTOLIC AND DIASTOLIC FUNCTION BY TISSUE DOPPLER IMAGING

Parameters	Period of Cardiac Cycle	Number of segments measured	Sampling Level
Systole			
Sa	Ejection period	Single or mean of 2 segments	Mitral septal or lateral annulus
Sm	Ejection period	Single or mean of 6 basal segments	Basal LV segments
Dyssynchrony	Time to peak or onset of Sm	Multiple, from 2 to 12 segments	Basal + mid segments
Diastole			
Ea	Early diastole	Single or mean of 2 segments	Mitral septal or lateral annulus
Em	Early diastole	Single or mean of 6 basal segments	Basal LV segments
Aa or Am	Late diastole	1 or 2 for Aa, up to 6 basal segments for Am	Mitral annulus for Aa, basal segments for Am
Ea/Aa or Em/Am	Diastole	1 or 2 for Aa, up to 6 basal segments for Am	Mitral septral or lateral annulus for Ea/Aa, basal segment for Em/Am
E/Ea or E/Em	Early diastole	Single or 2 locations	Mitral septral or lateral annulus for Ea/Aa, basal segment for Em/Am

Aa = mitral annular velocity during late diastole; Am = myocardial segmental velocity during late diastole; E/Ea = transmitral to mitral annular early diastolic velocity ratio; E/Em = transmitral to basal septal myocardial early diastolic velocity ratio; Ea = mitral annular velocity during early diastole; Ea/Aa = mitral annular early to late diastolic velocity ratio; Em = myocardial segmental velocity during early diastole; Em/Am = myocardial early to late diastolic velocity ratio; LV = left ventricular; Sa = mitral annular systolic velocity; Sm = myocardial segmental systolic velocity.

Tissue Doppler imaging has evolved as a new quantitative tool for the assessment of cardiac systolic function, diastolic function, and the hemodynamics of LV filling. From tissue Doppler velocity analysis, a number of parameters have been shown to be useful to predict long-term prognosis, in particular, Sm, Em, and E/Ea. The use of threshold value of E/Ea (≥15) has provided independent and incremental prognostic information in a number of

major cardiac diseases, such as HFand acute MI. In patients with suspected coronary heart disease, a high basal segmental Sm value (>6 cm/s) after dobutamine stress echocardiography is associated with lower mortality and MI and is superior to wall motion score. Although a number of post-processing techniques can be derived from TDI, such as strain and strain rate, the clinical utility of these modalities as prognosticators has not been established. Because tissue Doppler velocity imaging is readily available in most of the current echocardiographic systems, this information is now ready to apply toward optimal clinical management for patients who are vulnerable to the development of cardiovascular events.

CARDIOGENIC SHOCK

Cardiogenic shock is the most severe clinical expression of left ventricular failure and is associated with extensive damage to the left ventricular myocardium in more than 80 percent of STEMI patients in whom it occurs; the remainder have a mechanical defect such as ventricular septal or papillary muscle rupture or predominant right ventricular infarction. In the past, cardiogenic shock has been reported to occur in up to 20 percent of patients with STEMI, but estimates from recent large trials and observational databases report an incidence rate in the range of 7 percent. This low-output state is characterized by elevated ventricular filling pressures, low cardiac output, systemic hypotension, and evidence of vital organ hypoperfusion . Patients with cardiogenic shock caused by STEMI are more likely to be older, to have a history of a prior MI or congestive heart failure, and to have sustained an anterior infarction at the time of development of shock. The incidence of cardiogenic shock in patients with STEMI has been relatively stable since the mid-1970s, the short-term mortality rate has decreased from 70 to 80 percent in the 1970s to 50 to 60 percent in the 1990s. Cardiogenic shock is the cause of death in about 60 percent of patients dying after fibrinolysis for STEMI.

DIAGNOSIS.

Hemodynamic monitoring has been used extensively during the last decades for risk stratification and guiding treatment of patients with cardiovascular destabilization, especially in the scenario of acute heart failure and cardiogenic shock

Cardiogenic shock is characterised by

1)Marked & persistent (30 mts) hypotension with SBP < 90 mm Hg

2)Marked reduction of cardiac index (<1.8 L/min/m) in the face of

3)Elevated left ventricular filling pressure >18 mm Hg.

4) With signs of hypoperfusion –oliguria ,confusion & cold extremeties

Spurious estimates of left ventricular filling pressure based on measurements of the pulmonary artery wedge pressure can occur in the presence of marked mitral regurgitation, in which the tall V wave in the left atrial and pulmonary artery wedge pressure tracing elevates the mean pressure above left ventricular end-diastolic pressure. Accordingly, mitral regurgitation and other mechanical lesions such as ventricular septal defect, ventricular aneurysm, and pseudoaneurysm must be excluded before the diagnosis of cardiogenic shock caused by impairment of left ventricular function can be established. Mechanical complications should be suspected in any patient with STEMI in whom circulatory collapse occurs. It is important to exclude mechanical complications because primary therapy of such lesions usually requires immediate operative treatment with intervening support of the circulation by intraaortic balloon counterpulsation

PATHOLOGICAL FINDINGS.

At autopsy, more than two thirds of patients with cardiogenic shock demonstrate stenosis of 75 percent or more of the luminal diameter of all three major coronary vessels, usually including the left anterior descending coronary artery. Almost all patients with cardiogenic shock are found to have thrombotic occlusion of the artery supplying the major region of recent infarction, with loss of about 40 percent of the left ventricular mass. Patients who die as a consequence of cardiogenic shock often have “piecemeal” necrosis—that is, progressive myocardial necrosis from marginal extension of their infarct into an ischemic zone bordering on the infarction. This is generally associated with persistent elevation of cardiac biomarkers. Such extensions and focal lesions are probably in part the result of the shock state itself. Early deterioration in left ventricular function secondary to apparent extension of infarction may, in some cases, result from expansion of the necrotic zone of myocardium without actual extension of the necrotic process. Hydrodynamic forces that develop during ventricular systole can disrupt necrotic myocardial muscle bundles, with resultant expansion and thinning of the akinetic zone of myocardium, which in turn results in deterioration of overall left ventricular function.

Other causes of cardiogenic shock in patients with STEMI include mechanical defects such as rupture of the ventricular septum, a papillary muscle, or free wall with tamponade; right ventricular infarction; or marked reduction of preload caused by conditions such as hypovolemia**.**

PATHOPHYSIOLOGY - CLASSIC SHOCK PARADIGM

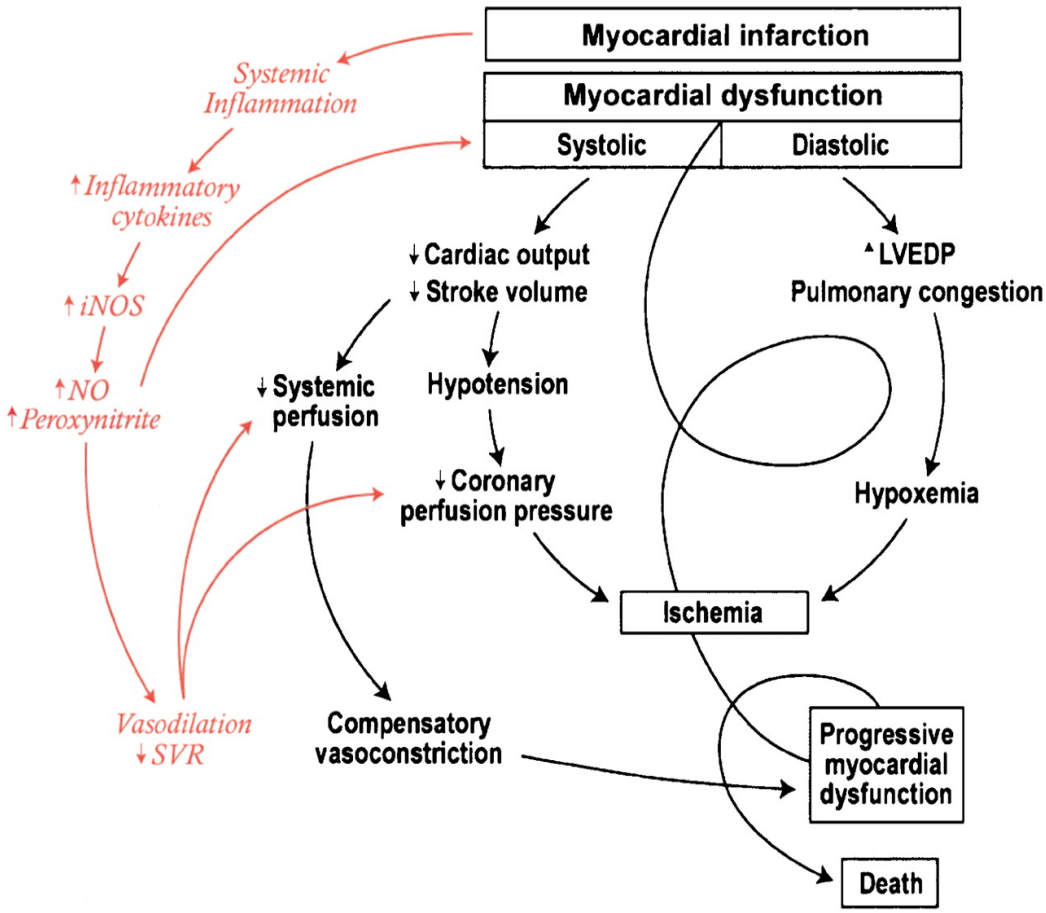


Figure 4 Classic shock paradigm, as illustrated by S. Hollenberg, is shown in black. The influence of the inflammatory response syndrome initiated by a large MI is illustrated in red. LVEDP indicates left ventricular end-diastolic pressure. .

MANAGEMENT

Of the five therapies frequently used to treat patients with cardiogenic shock (vasopressors, intraaortic balloon counterpulsation, fibrinolysis, PCI, and CABG), the first two are useful temporizing maneuvers. Surgical treatment in patients with cardiogenic shock aside from correcting mechanical abnormalities may involve bypassing occluded, as well as severely obstructed nonoccluded vessels. Occlusion of one major vessel can cause left ventricular dysfunction and hypotension, which can then lead to hypoperfusion and ischemia of myocardium subserved by the other diseased vessels. Left ventricular function can be improved by relief of this ischemia with revascularization.

Multiple observational series of patients undergoing balloon angioplasty in cardiogenic shock have demonstrated improved hemodynamic status and suggested enhanced survival. In contrast, thrombolysis appears less effective when administered to patients in shock. Although thrombolysis may provide a survival benefit for patients, increasing rates of PCI and declining rates of lytic use were associated with declining mortality for 25,311 shock patients in the National Registry of Myocardial Infarction (NRMI) database from 1995 to 2004. In this propensity-adjusted multivariable analysis, primary PCI was associated with a significant reduction in hospital mortality .

In the randomized SHOCK trial, a strategy of early revascularization resulted in 132 lives saved at 1 year per 1000 patients treated as compared with initial medical therapy followed by no or late revascularization as clinically determined.

The preferred treatment is PCI of the IRA for patients with 1- to 2-vessel coronary artery disease (CAD) and suitable lesions. Moderate 3-vessel disease, ie, 100% IRA occlusion, <90% stenosis in 2 other major vessels, or more severe lesions in second-order vessels, may be treated with PCI of the IRA and staged complete revascularization, as indicated. Glycoprotein IIb/IIIa

antagonists and stents are recommended. Immediate CABG is the preferred treatment for severe 3-vessel or left main CAD. If CABG cannot be performed, single-vessel or multivessel PCI may be attempted. Distal embolization in the non–IRA territories during PCI may be disproportionately harmful in the setting of shock or recent shock. Therefore, CABG is generally preferred to PCI when revascularization of the non–IRA artery is clinically indicated in the week after shock. However, early multivessel PCI may be warranted when shock persists despite PCI of the IRA, when CABG cannot be performed.

ECHOCARDIOGRAPHIC PREDICTORS OF SURVIVAL IN CARDIOGENIC SHOCK -SUB STUDY OF SHOCK TRIAL

From 1993 to 1998, 302 acute MI patients at 30 sites were randomized within 12 hours of cardiogenic shock diagnosis to either early emergency revascularization (ERV) or initial medical stabilization (IMS).

ECHOCARDIOGRAPHY

During the 4 years of the trial, 2D transthoracic echocardiograms were performed on each patient within 24 hours of randomization and predischarge The qualitative review included a complete evaluation of size and function of each ventricle and all valves in addition to LV regional wall motion assessment. A wall-motion score index was calculated by dividing the wall-motion score by number of segments visualized. Color Doppler of MR was graded with a 0 to 4 scale (0=none, 1=mild, 2=moderate, 3=moderate to severe, and 4= severe). Mitral valve leaflet geometry and morphology were assessed for presence of leaflet prolapse, flail, and incomplete closure.

Quantitative analysis included right and left atrial dimensions, LV dimensions, LV ejection fraction (EF), extent of LV endocardium involved by wall-motion abnormality, LV sphericity index, and mitral valve annular dimensions.. The sphericity index was calculated as a ratio of the LV midventricular dimension in the apical 4-chamber view and the long-axis

dimension. Doppler indices of LV filling were obtained from pulse-wave Doppler at the tips of the mitral valve and included E- and A-wave peak velocities, time velocity integrals, acceleration time, and deceleration time.

Of the early echocardiograms suitable for analysis, 82 were from the ERV group and 87 from the IMS patients. The characteristics of the patients with early echocardiograms were similar in terms of age, sex, rate of transfer admissions, and timing of shock and the echocardiograms.

The findings of the early echocardiogram showed mean LVEF was 31±11%. No significant differences in LV size or function were noted between the 2 groups. The wall-motion scores reflect significant regional dysfunction in both groups. No differences were observed between treatments. Regional function in the infarct zone was markedly impaired, with a mean segmental score approaching 3 representative of akinesis/dyskinesis.

MR of grades 2+ to 4+ was noted in 39.1% of patients. The degree of MR did not differ between treatment groups (ERV mean MR grade 1.4±0.9 versus IMS 1.3±0.9). Apical displacement of mitral leaflet coaptation, also known as incomplete mitral leaflet closure pattern was noted in 40% of those with grade 2+ to 4+ MR.

ECHOCARDIOGRAPHIC VARIABLES ASSOCIATED WITH SURVIVAL

The significant echocardiographic univariate predictors of 30-day survival were LVEF and severity of MR. These same variables, as well as end-diastolic and end-systolic LV volume, were univariate predictors of 1-year survival .An LVEF cutoff of 28% represents the median value. Furthermore, the separation of MR into those with less than grade 2+ and those with grade 2+ or higher provided the greatest discrimination between survivors and no survivors. From the early echocardiograms, the only independent multivariate predictors of either 30-day or 1-year mortality were MR severity (MR ≥2 versus

<2: 1-year odds ratio for death=6.64, *P*=0.0003) and LVEF (LVEF <28%: 1-year odds ratio for death 4.04, *P*=0.005).

PROGNOSIS AND DOPPLER ECHOCARDIOGRAPHY IN ACUTE CORONARY SYNDROME

Besides its usefulness for quantification of LV systolic function, Doppler echocardiography provides useful information for the assessment of diastolic function and LV filling pressures.

The *E*/*e*' ratio has been well validated to assess LV filling pressures .The threshold of *E*/*e*' > 15 identifies at best patients with mean LV diastolic pressures above 12 mmHg measured by micromanometer-tipped catheters.

Raised LV filling pressures indicate a relatively load intolerant myocardium. This may results from major myocardial damage due to coronary occlusion or conversely from minor damage associated with a previous stiff LV chamber due to aging, hypertension, diabetes, or coronary atherosclerosis. These patients with increased LV filling pressures show poor outcome.

The analysis of mitral inflow using pulsed Doppler signal recorded at the tips of the leaflets has a prognostic value in various cardiac diseases. Higher mitral *E*/*A* ratios and shorter deceleration times that define the restrictive pattern indicate an increased risk of adverse events after myocardial infarction.

Similarly *Temporelli et al.* have also observed a poor outcome in 571 patients enrolled in the GISSI-3 trial when mitral deceleration time is shortened. The propagation velocity of mitral inflow measured on M-mode colour Doppler echocardiography has also prognostic significance .However, reproducibility and quality of measurement of a colour M-mode slope that requires to carefully adjust the colour scale, in acute condition may be questionable in routine clinical practice.

By contrast, the direct recording of mitral annulus motion using tissue Doppler is easily obtained. The *E*/*e*' ratio gives a reasonable estimate of LV filling pressures and remains valid in the presence of sinus tachycardia, functional MR and preserved or depressed LV systolic function.

IMPORTANCE OF TISSUE DOPPLER-DERIVED DIASTOLIC FUNCTION IN STEMI PATIENTS

The data indicates that (i) bedside Doppler echocardiography obtained on admission provides prognostic information in patients with STEMI receiving a modern therapeutic strategy and (ii) the association between *E*/*e*' ratio >15 and the long-term risk of cardiac death was independent of clinical evidence of heart failure, as well as of renal dysfunction, blood glucose level, LV systolic dysfunction, and MR.

Bedside Doppler echocardiography provides additional prognostic information over clinical and biological parameters that are routinely determined in patients presenting with STEMI.

Current guidelines do not recommend index echocardiogram for patients admitted for unequivocal ACS. The present data advocate to perform index bedside Doppler echocardiography in the modern era of ACS management

STUDY DESIGN AND METHODS

Setting — Intensive Coronary Care Unit,
Department of Cardiology,
Govt Stanley Hospital,
Chennai-1.

Study design — Single centre, non randomized, observational and Prospective study

PATIENT SELECTION

The study population included 60 consecutive patients of which the population included: Group 1) 30 patients with chronic CHF as controls; and Group 2) 30 patients with CS.

The patients were of any age group and from both sexes admitted between May 2009 and May 2010 with a diagnosis of Acute STEMI with cardiogenic shock in coronary care unit (CCU) of our department. They were evaluated systematically for inclusion into study with history taking including risk factors, clinical examination and routine laboratory investigations including blood sugar,lipid profile, serum cardiac biomarkers ,12 lead ECG and bedside echocardiography. They were all included in the study after strictly adhering to study protocol.

All patients received standard clinical care including monitoring of vital functions in a coronary care unit during the initial hospital stay .All patients were given chewable Aspirin 300 mg, clopidogrel 300 mg. and atorvastatin. Eligible patients were thrombolysed with *Streptokinase* 1.5 million Units over 1 h and inotropic agents dopamine & dobutamine where used.

The study was approved by the local institutional ethical committee

INCLUSION CRITERIA

Group 1

Patients were included in this group if they fulfilled the following criteria

- The CHF group was defined as LVEF < 40%, NYHA class III or IV status, requiring hospitalization for decompensated heart failure.

- The cause of their cardiac failure should be coronary artery disease in the form of prior admission for anterior wall myocardial infarction.

Group 2

Patients were included in this group if they fulfilled the following criteria

-Patients with Acute Anterior wall STEMI - with cardiogenic shock.

Cardiogenic shock was diagnosed if the patient had hypotension - a systolic blood pressure < 90 mmHg for at least 30 min or the need for supportive measures to maintain a systolic blood pressure ≥ 90 mmHg .

EXCLUSION CRITERIA

-Preexisting severe co morbid conditions which may influence or modify the clinical course and outcome which may also preclude acquisition of adequate echocardiographic and other data.

CKD

Cor Pulmonale

Chronic Liver disese

-NON STEMI

- Atrial fibrillation

-Second and third degree AV Block.

-Evidence of primary myocardial or

-Valvular heart disease

- Ventricular septal rupture

- Ventricular free wall rupture and

- Acute mitral regurgitation with papillary muscle rupture

- Prior Angioplasty

-Proir CABG

CLINICAL DATA

Death was defined as all-cause mortality during hospitalization.

Vital signs and Killip class findings were collected at the time of hospital presentation.

Killip class I was defined as the absence of congestive heart failure that is patients who are free of rales and a third heart sound (S_3).

Class II patients have rales but to only a mild-moderate degree (<50 percent of lung fields) and may or may not have an S_3 and or jugular venous distention,

Class III have frank pulmonary edema and

Class IV as cardiogenic shock.

Patients with cardiogenic shock were identified.

Cardiogenic shock was defined as systolic blood pressure <90 mm Hg for 30 min and a urine output of less than 20 ml/hr in the setting of an acute ischemic insult. Patients in whom systolic blood pressure increased to >90 mm Hg within 1h after administration of positive inotropic agents, or patients who died within 1h of hypotension but met other criteria for cardiogenic shock, were still classified as having cardiogenic shock.

ELECTROCARDIOGRAPHIC DATA

STEMI was diagnosed based on following ECG Criteria and STEMI was later confirmed by the elevation of cardiac enzymes with CK-MB.

ST-segment elevation of at least 0.1 mV in two or more contiguous limb leads;

At least 0.2 mV in two or more contiguous precordial leads

Standard 12-lead electrocardiograms (ECGs) were collected at baseline and 90 minutes of starting streptokinase treatment, then every 24 hours until discharge.

Reinfarction was defined as re elevation of CK-MB above the upper limit of normal and increase by at least 50% of the previous value or by the presence of new Q waves in the ECG.

ECHOCARDIOGRAPHY

2D transthoracic ECHO and Tissue Doppler imaging were performed on each patient at the time of admission and repeated and or after hemodynamic instability. The echocardiograms were analyzed for qualitative and quantitative assessment. The assessments were performed with using an Aloka SSD 4000 phased array system equipped with tissue doppler and harmonic imaging technology with Doppler frequency of 2.5 to 3.8 mHz..

The qualitative review included a complete evaluation of size and function of each ventricle and all valves in addition to LV regional wall motion assessment.

Regional wall motion was analyzed individually on the basis of its motion and systolic thickening. The segments were analyzed as either Hyperkinetic/Normal/MildHypokinesis/Hypokinesis/Severehypokinesis/ Akinesis and Dyskinesis (paradoxical systolic motion)/ Aneurysmal .

The severity of mitral regurgitation (MR) was semi-quantitatively graded from color-flow Doppler images in the apical four- and two-chamber views .Color Doppler of MR was graded with a 0 to 4 scale (0=none, 1=mild, 2=moderate, 3=moderate to severe, and 4= severe).

The severity of MR was classified as

Mild (jet area/left atrial area, 20% in the absence of wall jet),

Moderate (jet area /left atrial area 20–40%),

Severe (jet area/left atrial area >40%)

Mitral valve leaflet geometry and morphology were assessed for presence of leaflet prolapse, flail, and incomplete closure.

Quantitative analysis was then performed on echocardiograms of sufficient quality. Measurements included LV dimensions, LV volume, LV ejection fraction (EF) .

LVEF was measured by M- MODE, Modified Simpson's method

LVEF was stratified as

Normal (LVEF 55%),

Mildly reduced (45–54%),

Moderately reduced (30–44%),

Severely reduced (< 30%)

Patients who had mechanical complications such as VSR, acute mitral , regurgitation with papillary muscle rupture and free wall rupture were excluded

Diastolic dysfunction, pericardial effusion, LV thrombus, tricuspid regurgitation and pulmonary hypertension were noted

Pulsed Doppler mitral inflow velocities were obtained by placing a 1–2 mm sample volume between the tips of the mitral leaflets in the apical four-chamber view. Spectral gain and wall filter settings were optimized to clearly display the onset and cessation of LV inflow .The Doppler beam was aligned parallel to the direction of flow. Spectral mitral velocity recordings were initially obtained at sweep speeds of 25 to 50 mm/s for the evaluation of respiratory variation of flow velocities in patients with pulmonary or pericardial disease. If variation is not present, the sweep speed was increased to 100 mm/s.

The following variables were measured in end expiratory:

Peak early filling velocity (E)

Peak filling velocity at atrial contraction (A velocity)

E/A ratio

Deceleration time of the peak E velocity, defined as the slope from peak E extrapolated to the baseline value

IVRT

Restrictive mitral inflow patterns were defined as E/A ratio >2 with a E-wave deceleration time < 160 ms.

TISSUE DOPPLER IMAGING

TDI of the mitral annulus was obtained from the apical 4-chamber view.

A 1.5-mm sample volume was placed at or 1 cm of the lateral and medial mitral annulus.

Analysis was performed for the early diastolic (E') ,late diastolic velocity (A')and peak systolic velocity (S'). These variables were analyzed individually, as the average of the medial and lateral annulus.

All Doppler signals were recorded with at 100 mm/s.

The average of 3 end-expiratory cycles was used.

E/E' Ratio was calculated.

Mitral septal annular systolic(S') velocity cut off value of <7.5 cm/s was taken as LV dysfunction.

RESULTS AND DATA ANALYSIS

Statistics

The data are summarized as mean ± SD or number (percentage). Chi-square or Fisher exact tests were applied to compare categorical variables. A Student t-testing was used to compare parameters between both groups. A p value < 0.05 was considered significant. The SPSS Statistical Analysis System was used to perform the analysis.

Baseline characteristics

The total population included 60 patients (mean age 59 ± 11, range 45 to 72). Group I consisted of 30 patients (mean age 58 ± 13 years, range 45 to 69) with chronic CHF as controls. Group II consisted of 30 patients (mean age 60 ± 10 years, range 46 to 72) with CS.The sex distribution in the study groups are as follows. total population 60 patients – 51 males & 9 females . Group I consisted of 30 patients -25 males (83%) & 5 females (17%) . Group II consisted of 30 patients -26 males(86%) & 4 females (14%).

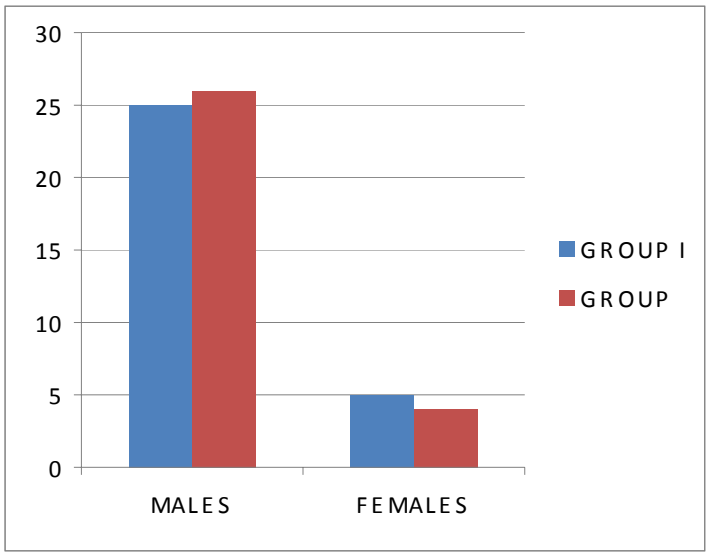


Diagram 1 - Sex distribution

The occurrence of Diabetes Mellitus in the study groups are as follows Group I 18 out of 30 patients (60%) and in Group II 20out of 30 patients (66%) had Diabetes Mellitus.

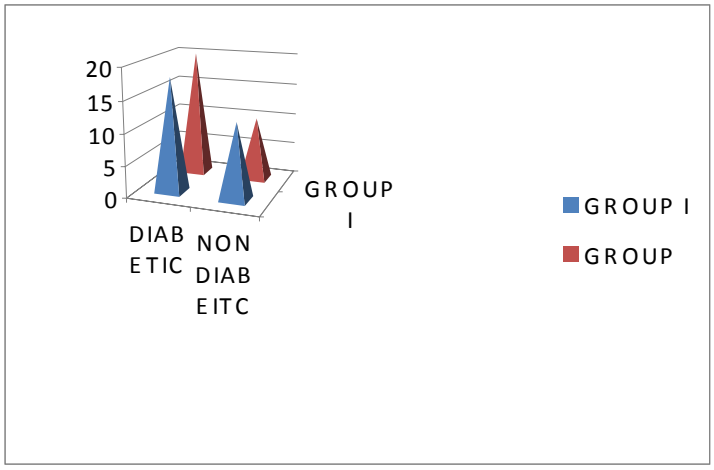


Diagram 2 – Diabetic population

The occurrence of Hypertension in the study groups are as follows Group I 21 out of 30 patients (71%) and in Group II 23out of 30 patients (75%) had HT.

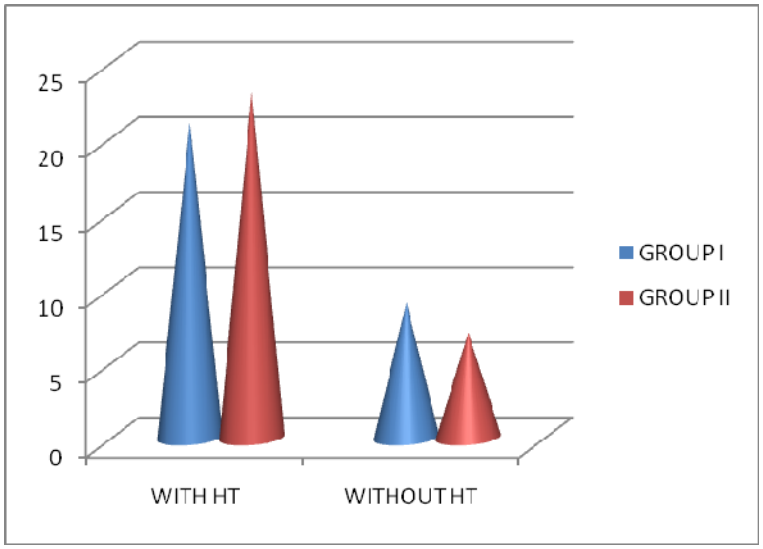


Diagram 3 - Hypertensive population

The occurrence of Dyslipidemia in the study groups are as follows Group I 14 out of 30 patients (47%) and in Group II 16 out of 30 patients (53%) had Dyslipidemia

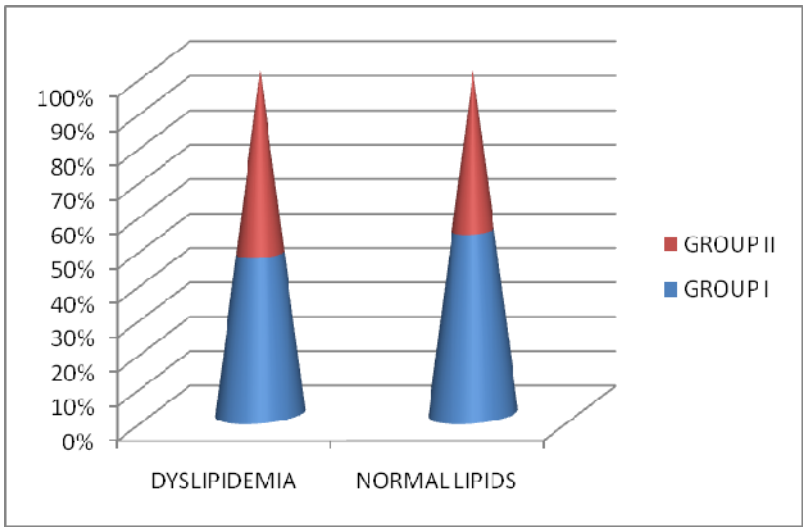


Diagram 4 – Dyslipidemic population

The occurrence of Smoking history in the study groups are as follows Group I 21 out of 30 patients (70%) and in Group II 19 out of 30 patients (63%) had history of smoking.

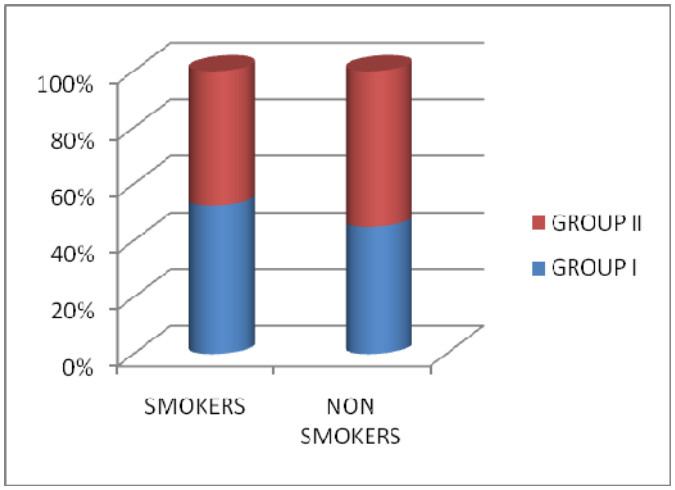


Diagram – 5 Smoking

The in-hospital mortality in the total study population are as follows Group I 4 out of 30 patients (13%) and in Group II 19 out of 30 patients (63%).

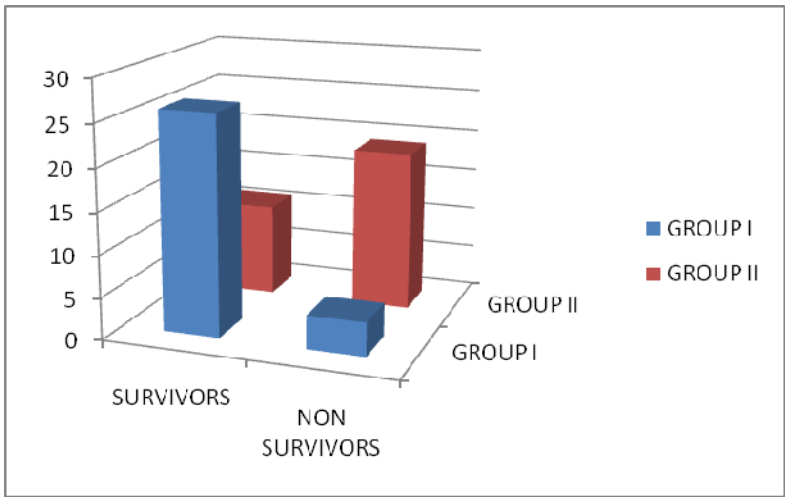


Diagram 6 – Mortality

Baseline characteristics are shown in Table 5. The two groups were similar with respect to baseline demographics and cardiovascular risk factors.

Table 5

Clinical findings in all patients (n = 60)			
Characteristics	**CHF (n = 30)**	**CS (n = 30)**	**p-value**
Age (y)	58 ± 13	60 ± 10	0.85
Male Gender (%)	25 (83)	26 (86)	0.92
Diabetes mellitus (%)	18 (60)	20(66)	0.80
Hypertension (%)	21 (71)	23 (75)	0.78
Dyslipidemia (%)	14 (47)	16 (53)	0.82
Smoking history (%)	21 (70)	19 (63)	0.81

Values are mean ± SD (percentage). CHF, congestive heart failure; CS, cardiogenic shock; y, years; LVEDD, EF, ejection fraction. $p < 0.05$* was considered significant.

Despite the difference in concomitant medication use amongst both groups; with all patients in the CS group being on inotropic support, the HR, SBP and DBP were similar for assessment of conventional diastolic and TDI parameters .

The mean duration of hospital stay of the study population was 10 ± 2 days.

All patients in Group I had a past history of AWMI and thrombolysis with streptokinase and none in Group II had a prior MI.

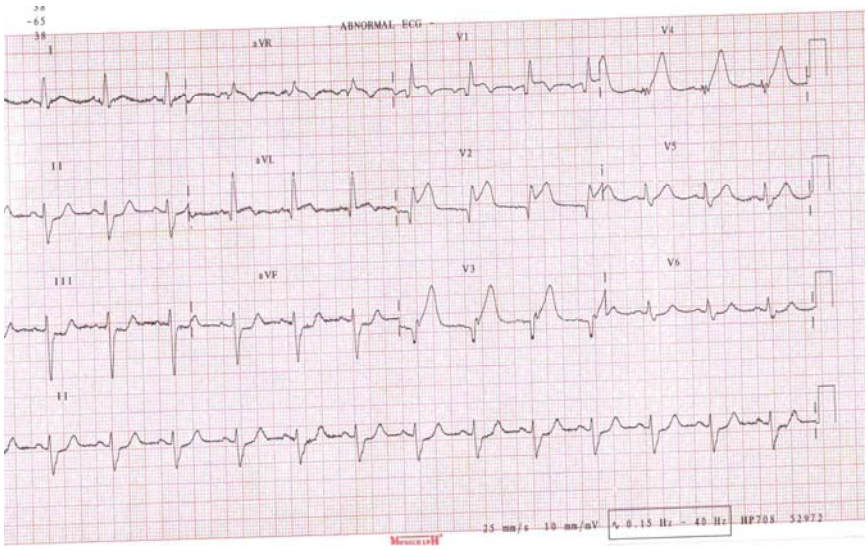


Figure 5 ECG of a patient showing AWMI

The in-hospital mortality in the CHF cohort was 13% as compared to the CS group with an in hospital mortality of 63% and most of them were males , 3 out of 4 (75%) in Group I and 18 out of 19 (95%) in Group II.

Echocardiographic analysis

Table .6 Echocardiographic variables

2-D echocardiographic and Doppler echocardiographic findings in all patients (n = 60)			
Characteristics	**CHF (n = 30)**	**CS (n = 30)**	**p-value**
LVEDD (mm)	60 ± 3	58 ± 3	0.82
EF (%)	27± 3	25± 5	0.80
Doppler echocardiography			
Mitral E velocity (cm/s)	70± 9	71 ± 7	0.73
Mitral A velocity (cm/s)	63 ± 12	65 ± 11	0.71
E/A ratio	1.02 ± 0.2	1.03 ± 0.3	0.77
Doppler tissue imaging			
S' (cm/s)	5 ± 1	3.0 ± 0.8	< 0.01
E' (cm/s)	5.0 ± 1.0	3.1± 1.1	< 0.01
A' (cm/s)	3.7 ± 1.0	3.1 ± 1.0	0.92
E/E'	13 ± 3	21 ± 2	< 0.01

Values are mean ± SD (percentage). CHF, congestive heart failure; CS, cardiogenic shock; $p < 0.05$* was considered significant

The two groups were similar with respect to 2D echocardiographic parameters. Of the entire cohort, the mean LVEF was 25 ± 5%.

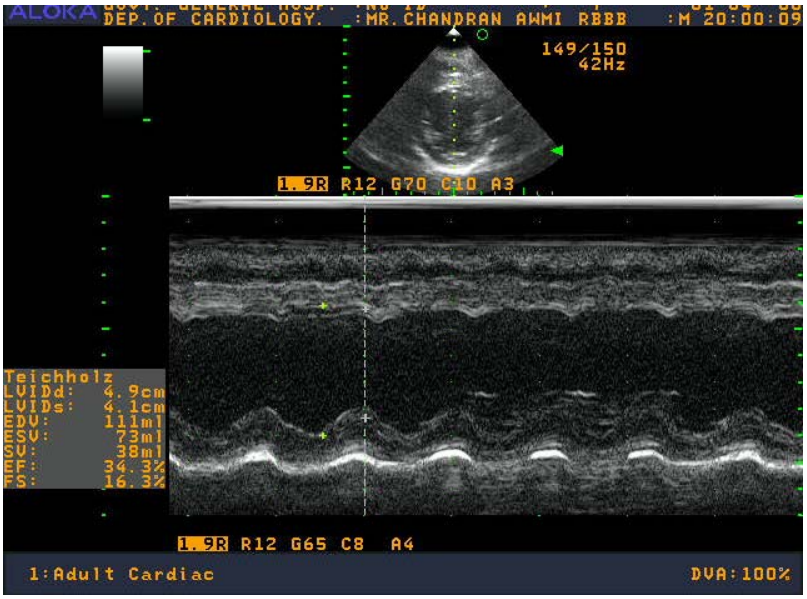


Figure 6 M-Mode Echo- of a patient with AWMI showing hypokinesis of IVS with compensatory hyperkinesis of LVPW.

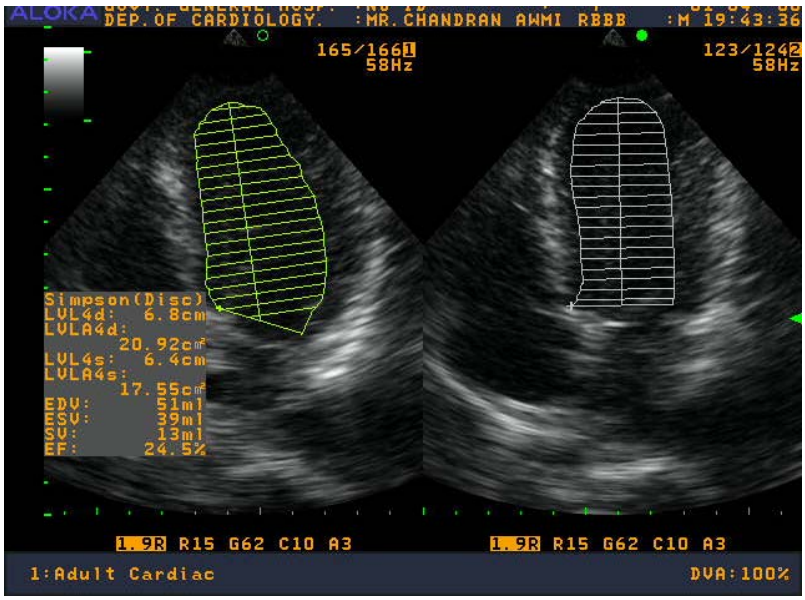


Figure 7 2D echo–apical 4 chamber view. LVEF assessment by modified Simpson's method.

The incidence of significant MR (Grade ≥ 2+)was present in 16% in Group I (5 out of 30) and 13% in Group II (4 out of 30) . There was no significant difference in mild MR (Grade < 2+) between the two groups (7out of 30)in Group I & (6 out of 30) in Group II. The toal (Group I & Group II) incidence of MR in the mortality group was 66 % (14 out of 21).

All 60 patients had abnormal diastolic function, either having a pseudonormal pattern or a delayed relaxation pattern or a restrictive filling pattern based on conventional diastolic parameters.Most of the survivors had either pseudonormal pattern or a delayed relaxation pattern and most of the non survivors had restrictive filling pattern .

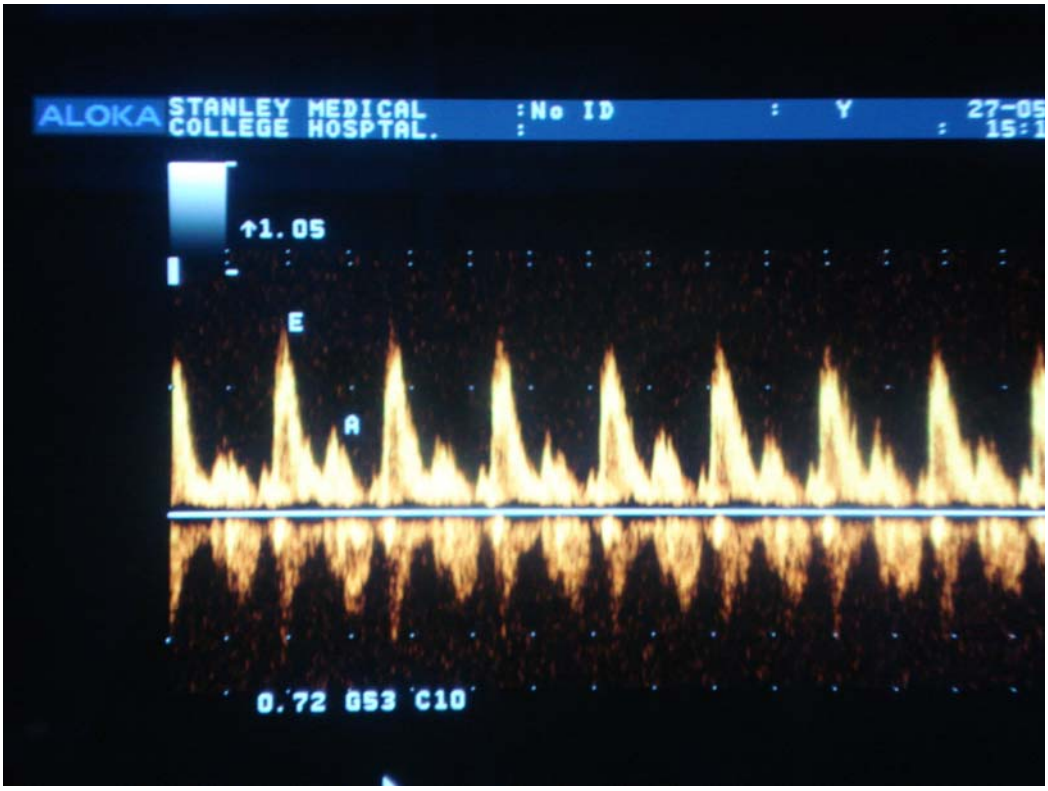


Figure 8 Pulsed wave Doppler echo of mitral inflow showing shortened DT with restrictive filling pattern in a patient with AWMI who developed Cardiogenic Shock

The Doppler Echo indices namely Earlydiastolic mitral inflow velocity E, Late diastolic mitral inflow velocity and E/A ratio showed no statistical significant difference between the two groups - Table 6.

Majority of patients in the survival group had Mitral deceleration time (DT) of >160 ms where as DT of < 160 ms was associated with increased mortality .

IVRT greater than 90 ms was observed in majority of the survival group and IVRT less than 70 ms was observed in most of the non survival group.

The mitral annular Systolic velocity (S') was significantly lower in Group II patients when compared to patients in Group I. ie 3.0 $\pm$ 0.8 cm/s Vs 5.0 $\pm$ 1cm /s . In Group II patients mitral annular Systolic (S') was higher in the survivor group when compared to those patients who died of cardiogenic shock. ie 3 $\pm$ 0.9 Vs 2 $\pm$ 0.5 cm/s

The mitral annular early diastolic (E') velocity was significantly lower in Group II patients when compared to patients in Group I. ie 3.1 $\pm$ 1.1 Vs 5.0 $\pm$ 1.0 cm/s . In Group II patients mitral annular early diastolic (E') velocity was lower in the non-survivor group .

The E/E' ratio was elevated in CS patients as compared to CHF patients.ie 21 $\pm$ 2 Vs 13 $\pm$ 3 .In Group II patients E/E' ratio was higher in the mortality cohort when compared to the survivor group .

Restrictive mitral inflow pattern and mitral E/E' ratio >15 was more frequent in the mortality group.

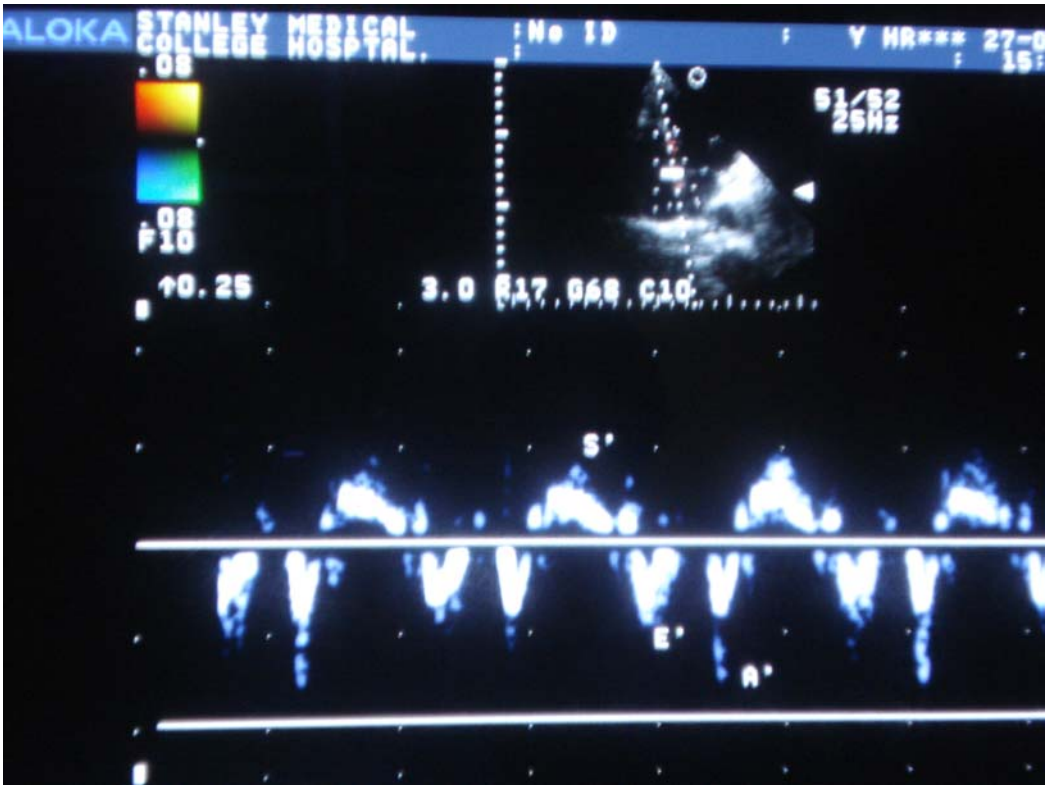


Figure 9 TDI of the same patient as in figure 9 showing reduced E' value with raised E/E' ratio of > 15 .The S' value is also reduced suggesting LV dysfunction .

DISCUSSION

Cardiogenic shock is the leading cause of death in patients with acute myocardial infarction with an in-hospital mortality rate greater than 50% to 60 % [10] in the current era when compared to 70 % to 80 % in the 1970 ‘s. A variety of structural and functional abnormalities are identifiable on echocardiography in patients presenting with acute CS [11].

Although *Picard et al* demonstrated reduced LV systolic function and mitral regurgitation as predictors of survival and response to early revascularization in CS [11], this study shows a reduction in TDI derived indices in CS patients. Despite similar reductions in LV systolic function, this study demonstrated CS patients have reduced myocardial velocities and higher filling pressures using TDI as compared to CHF patients.

Mitral annular systolic velocity (S') reflects the long axis motion of the ventricle which is an important component of LV systolic function. Peak myocardial systolic velocity averaged from six sites around the mitral annulus correlates well with LV ejection fraction.

A S' value greater than 7.5 cm/s had a sensitivity of 79% and a specificity of 88% in predicting normal global LV function [42].

Sub-endocardial fibers make a substantial contribution to long axis function, and are susceptible to a variety of cardiac pathologies. Hypertension, coronary artery disease and CHF have been shown to negatively affect these fibers and reduce S'. A reduction in S' correlates with increased morbidity and mortality in each of these disease states [43 ,41,32].

In this study, CS patients had a significantly lower S' compared to CHF patients. Shock has severe metabolic consequences, involving mainly energy metabolism, substrate utilization, and acid-base regulation. As longitudinally

arranged subendocardial fibers are most vulnerable to ischemia and metabolic abnormalities, it is not an unexpected finding that LV base-apex contraction was abnormal in this CS population. Given the prognostic value in other cardiac disease states, a reduction of S' in CS may be of similar clinical importance in this patient population. In this study, albeit small numbers, the subset of patients in CS who survived demonstrated increased S' values on initial presentation, as compared to those who died in-hospital.

Similar to systolic annular velocity (S'), early diastolic mitral annular (E') appears to be a strong independent predictor for the short and long-term prognosis of patients with cardiovascular disease.

E' has been proposed as a useful index for the non-invasive evaluation of LV relaxation, that is relatively preload independent [17]. Recent studies have shown that a reduced E' predicts increased cardiovascular morbidity and mortality [43,13,32].

Wang et al demonstrated that an E' less than 3 cm/s was the best prognostic marker for long-term follow up in a population of patients with chronic hypertension [43].

In the current study, E' was significantly reduced in the CS population as compared to chronic CHF patients. The potential mechanism for this abnormality may relate to the acute increase in LV wall stress due to the ischemic insult in the CS state. The reduced E' may be a sensitive marker of regional relaxation abnormality reflected in the long axis dimension that could potentially be evident earlier than clinical manifestations of global left ventricular relaxation abnormality.

Raised LV filling pressures indicate a relatively load intolerant myocardium. This may result from major myocardial damage due to coronary

occlusion or conversely from minor damage associated with a previous stiff LV chamber due to aging, hypertension, diabetes, or coronary atherosclerosis

Since E' is reduced and diastolic mitral annular increases with higher filling pressures, the E/E' ratio correlates well with invasive pulmonary capillary wedge pressure (PCWP) measurements [37,17,26]. Elevated PCWP is associated with a higher mortality rate after acute MI and has been shown to carry independent prognostic information in CHF patients [12,30].

The E/E’ ratio has been well validated to assess LV filling pressures. The threshold of E/ E’ >15 identifies at best patients with mean LV diastolic pressures above 12 mmHg measured by micromanometer-tipped catheters.

Doppler echocardiography provides useful information regarding LV filling pressure. *Ommen et al*. have previously reported that ratio of early transmitral flow (E) to early mitral annulus velocities (E’) >15 indicates elevated LV end diastolic pressure measured by cardiac catheterization and associated with poor prognosis. Other investigators have observed a poor outcome in a retrospective cohort of patients with ACS when E/ E’ ratio is >15.

These patients with increased LV filling pressures show poor outcome in this study .In addition, the direct recording of mitral annulus motion using tissue Doppler is easily obtained. The E/ E’ ratio gives a reasonable estimate of LV filling pressures and remains valid in the presence of sinus tachycardia, functional MR, and preserved or depressed LV systolic function.

In this study, CS patients had an elevated E/E' ratio confirming raised LV filling pressures. The spectrum of diastolic abnormalities which may account for the elevated ratios include increased myocardial stiffness, reduced LV compliance, and elevated LV end-diastolic parameters. A restricted filling pattern tends to correlate with higher mortality at 30 days and 1 year in CS [12], and is known to be associated with a poor prognosis in CHF patients [32].

Hillis et al. previously established that in the setting of acute MI, an E/E' ratio greater than 15 can identify patients at an increased risk of mortality with a sensitivity of 70% and a specificity of 91% [30,29]. In addition, the prognostic value of E/E' was incremental to clinical factors and conventional echocardiographic parameters of LV systolic and diastolic function [30,25]. An elevated E/E' ratio in CS patients may be of comparable value in distinguishing this select population at higher risk for poor cardiovascular outcomes [8].

The present data indicate that the association between E/E' ratio >15 and high mortality and morbidity independent of clinical evidence of heart failure, LV systolic dysfunction and MR.

The E/E' ratio is superior to conventional parameters of LV systolic function, such as LVEF for prediction of prognosis. However, it is important to recognize that measurement of E/E' provides complementary prognostic data, with the maximum information obtained by combining this with clinical, systolic, and conventional diastolic parameters.

Limitations

Similar to other studies using TDI, this methodology is affected by the quality of 2D images and cardiac translation, rotation, or both.

The reduced LV systolic function in the CS population may have represented myocardial stunning, and thus the true LVEF may have been underestimated in this subgroup.

Tissue Doppler imaging parameters was not obtained immediately following revascularization nor at 6 month followup in the patient population.

Lastly, this study is limited by the relatively small sample size and a larger, prospective study is needed in order to make more substantive

conclusions regarding the clinical utility of tissue Doppler indices prior to percutaneous revascularization in patients with cardiogenic shock

CONCLUSION

-Besides Bedside 2-D and Doppler echocardiography, TDI provides additional prognostic information over clinical and biological parameters that are routinely determined in patients presenting with STEMI- CS

-Systolic annular velocity (S') correlates with LV systolic function.

-A Systolic annular velocity S' value greater than 7.5 cm/s predicts normal global LV function.

-A reduction of systolic annular velocity (S') occurs both in patients with CHF and in patients with CS .

-The reduction of Systolic annular velocity (S') is greater in patients with CS than in patients with CHF.

-Patients who have survived of CS demonstrate comparatively increased systolic annular velocity (S') values than those who died in -hospital.

- Mitral annular early diastolic velocity E' was significantly reduced in the CS population as compared to chronic CHF patients

- The reduced E' is a sensitive marker of regional relaxation abnormality reflected in the long axis dimension

-E' gets reduced and diastolic mitral annular increases with higher filling pressures and the E/E' ratio correlates well with raised LV filling pressures.

- E/E' ratio in the setting of acute MI -CS greater than 15 can identify patients at an increased risk of mortality.

-The prognostic value of E/E' was incremental to clinical factors and conventional echocardiographic parameters of LV systolic and diastolic function An elevated E/E' ratio in CS patients may be of comparable value in distinguishing this select population at higher risk for poor cardiovascular outcomes.

- So this study shows a higher reduction of TDI indices namely S' , E' and A' in patients with CHF.

Current guidelines do not recommend index echocardiogram for patients admitted for unequivocal AMI. But recent studies advocate to perform index bedside Doppler echocardiography especially, Tissue Doppler Imaging in the modern era of AMI management.

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Cardiovascular Ultrasound 2008, 6**:**11doi:10.1186/1476-7120-6-11

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.

PROFORMA

NAME: AGE/SEX: CD.NO: DOA:

ADDRESS: OCCUPATION:

HISTORY: PRESENT PAST

RISK FACTORS: DM: HT: SMOKING: DYSLIPIDEMIA:

O/E: PR: BP: CVS: RS:

OTHER SYSTEMS:

DIAGNOSIS:

ECG

BLOOD INVESTIGATIONS : BASELINE RFT LIPID PROFILE

BLOOD SUGAR CARDIAC ENZYMES

TREATMENT GIVEN:

ECHOCARDIOGRAPHY:

GLOSSARY

CAD	Coronary Artery Disease
ACS	Acute Coronary Syndrome
AMI	Acute Myocardial Infarction
STEMI	ST Elevation Myocardial Infarction
CS	Cardiogenic Shock
CCU	Coronary Care Unit
CABG	Coronary Artery Bypass Graft
IVRT	Isovolumic Relaxation Time
MR	Mitral Regurgitation
SHOCK	Should we emergently revascularize Occluded Coronaries in Cardiogenic Shock?
PCWP	Pulmonary Capillary Wedge Pressure
PW	Pulse Wave
PNF	Pseudonormal Filling
TDI	Tissue Doppler Imaging
DT	Deceleration Time
Ea	Early diastolic velocity at mitral annulus
EF	Ejection Fraction
HF	Heart failure
LV	Left ventricle
LVEF	Left ventricular ejection fraction
Sa	Peak systolic velocity at mitral annulus
Aa	Late diastolic velocity at mitral annulus
Em	Early diastolic velocity at myocardial segments
Sm	Peak systolic velocity at myocardial segments

MASTER CHART GROUP II

S.No.	Name	Age	Sex	C.D.No.	DM	HT	Dys	Smoking	LVIDd mm	LVEF %	MR	E cm/s	A cm/s	E/A	S' cm/s	E' cm/s	A 'cm/s	E/E'	Survivors
1	John	60	M	186160	Y	Y	N	Y	58	25	N	66	70	0.9	3	3.6	3.1	18.3	
2	Kalim	70	M	18224	Y	N	Y	Y	57	20	N	78	56	1.3	2.9	3.3	3	23.6	Died
3	Chockalingam	64	M	185402	N	Y	N	Y	59	21	<2	77	60	1.2	2.8	3.7	2.2	20	Died
4	Kannammal	70	F	185384	Y	Y	Y	N	50	25	N	66	54	1.2	3.1	3	3.4	22	
5	Sowki fasman	50	M	14987/09	Y	Y	Y	Y	55	27	N	67	70	0.9	3	3.3	3.5	20.3	
6	Annamalai	52	M	185412	N	N	N	Y	60	24	<2	75	72	1	2.8	3.5	2.2	21.4	Died
7	Sambasivam	55	M	185330	Y	Y	Y	Y	57	30	N	64	76	0.8	3.2	3.6	4	17.7	
8	Kamala	65	F	187338	Y	Y	Y	N	59	25	≥2	78	58	1.3	2.2	3.4	2.5	22.9	Died
9	Durai	60	M	187325	N	Y	N	Y	58	24	N	76	70	1	2.3	3.3	2.7	23	Died
10	Loganathan	59	M	185335	Y	N	N	N	55	29	N	64	70	0.9	3.4	3.7	3.2	17.29	
11	Sivananthan	65	M	188174	Y	Y	Y	Y	55	24	<2	76	69	1.1	24	3.9	3	19	Died
12	Kokila	63	F	18565	N	Y	N	N	56	30	N	66	67	0.9	3.8	3.8	3.1	17.36	
13	Veerappan	63	M	28028	Y	Y	Y	N	56	22	N	78	70	1.1	2.5	3.8	3.1	20	Died
14	Ismail	60	M	190756	Y	N	N	Y	58	21	≥2	77	66	1.1	2.5	3.7	4	20.8	Died
15	Sowry	50	M	167550	N	Y	N	Y	60	27	N	65	70	0.9	3.7	3.9	2.9	16	
16	Durairaj	62	M	38993	Y	Y	Y	N	59	23	N	78	70	1.1	2.6	3.9	3.3	20	Died
17	Shantha kumar	52	M	185356	Y	N	N	Y	60	28	N	64	56	0.9	3.8	4.1	2.9	15	
18	Thirupandeeran	65	M	192606	N	Y	N	Y	59	20	<2	77	68	1.1	2.3	3.8	3.1	20.2	Died
19	S.K.Rao	70	M	195112	Y	Y	Y	N	55	21	N	76	69	1.1	2.2	4	4	19	Died
20	Daisy	65	F	185406	Y	Y	Y	N	60	28	N	65	67	0.9	3.7	4.1	3	15.8	
21	Sivananthan	65	M	188174	Y	Y	Y	Y	57	22	N	75	54	1.3	2.7	3.2	2.9	23	Died
22	Sounderaju	50	M	20752	N	N	N	Y	61	29	N	65	66	0.9	3.6	4	4.1	16.25	
23	Siddharth	52	M	7946/10	N	Y	N	Y	59	25	≥2	77	61	1.2	2.9	3.9	3	19.7	Died
24	Karunanithi	46	M	188174	Y	Y	Y	Y	56	24	N	78	69	1.1	2.6	3.4	3	22	Died
25	Mani	50	M	185312	Y	Y	Y	N	55	25	<2	76	68	1.1	2.5	3.7	3.5	20.5	Died
26	Dhavaman	70	M	235804	N	Y	N	Y	59	20	N	70	71	0.9	2.9	3.8	3.7	18	Died
27	Kesavan	72	M	190004	Y	Y	Y	N	58	23	≥2	75	66	1.1	2.4	3.1	2.7	24	Died
28	Mahimraj	46	M	10855	Y	Y	Y	Y	57	25	N	78	59	1.3	2.7	4	2.5	19.5	Died
29	Abdul rehuman	57	M	27190/10	N	Y	N	Y	60	27	N	66	70	0.9	3.1	3.9	3	16	
30	Balakrishnan	65	M	185343	Y	Y	Y	N	58	24	<2	66	67	0.9	2.2	3.6	2.4	18.3	Died

Y= YES ,N=NO